Volume 46 | Issue 1 Article 4

1984

Neonatal Calf Care

J. Todd Minor
Iowa State University

Richard L. Riese
Iowa State University

Neonatal Calf Care

J. Todd Minor, BS*
Richard L. Riese, DVM**

Introduction

Calf mortality is important economically in dairy and beef herds. Calf losses substantially reduce the returns from cattle raising. The economic losses are direct and indirect. Direct losses are those derived from the loss of the calf, planning and labor already invested by the herdsman, and veterinary expenses from obstetric and post-natal care. Indirect losses are infertility, chronic disease, and impaired development of the calf.

The occurrence rate of calf mortality depends on many factors: breed, farm management, farm climate and locale, age of the dam, lactation number of the dam, the bull, the duration of the pregnancy, and sex of the calf.[1] Calf mortality appears to be similar in beef and dairy cattle. Perinatal deaths are very important. In some European countries this accounts for 50–60% of mortality in calves born to heifers. Dystocia associated with large calves is also a significant cause of perinatal death. There is a close relationship between problem births and perinatal mortality.[1] The increasing use of large breeds, earlier calving, selection for growth rate, size, and production, and more intensive management systems has increased the incidence of dystocia and calf mortality.

The future of the cattle industry is based on the birth of healthy calves. With the advent of embryo transfer, the value of many of these calves has greatly increased. The death of any calf in the periparturient period is becoming a loss of greater importance to the herdsman. This paper presents some of the advice that can be given and the steps that a veterinarian can take to limit the losses of newborn calves. Preparturient care of the dam, problems arising from congenital malformations, and techniques for relief of dystocia will not be discussed.

*Mr. Minor is a fourth-year student in the College of Veterinary Medicine at Iowa State University.

**Dr. Riese is an assistant professor of Veterinary Clinical Sciences at Iowa State University.

Delivery

A normal full-term fetus' survival depends in large part on its dam's ability to deliver it quickly. The fetal factors exerting the greatest influence on parturition are sex, birth weight, and presentation. The fetus determines when it is born. The dam's influences are chiefly feto-pelvic compatibility and parity.[2]

Upon natural delivery of a healthy calf, the following calf blood gas and acid-base parameters have been found:[3]

pCO_2 = 58.15 mm Hg
pO_2 = 25.34 mm Hg
pH = 7.245
Base deficit = 3.86 mmole/L

Upon delivery of a healthy calf by caesarian section, the following calf blood gas and acid-base parameters have been found:[4]

pCO_2 = 60.7 mm Hg
pO_2 = 19.0 mm Hg
pH = 7.242
Base deficit = 3.13 mmole/L

All calves immediately after birth have a slight combined respiratory and metabolic acidosis. The metabolic part is compensated for by the calves in one to two hours. The respiratory portion lasts 24–48 hours.[3,4,5]

This data on normal calves will contrast sharply with the data from calves suffering from dystocia and calves delivered by caesarian section before physiologic parturition has begun. Delayed passage through the birth canal with placental disruption and/or caruncle compression compromises oxygenation. The oxygen transport via uterus/placenta/umbilical vein/fetal pathway is disturbed causing fetal hypoxia to anoxia and acidosis. One sign that this may have occurred is finding the neonate stained with meconium. Hypoxic stress in utero may result in the anal sphincter relaxing and hyperperistalsis. Meconium voided into the amniotic fluid stains the fetus. The neonate can breathe with its nose past the labia of the dam, but it can't expand its chest in the narrow birth canal. The oxygenation compromise is aggravated by continuous traction.

Calves subject to asphyxia in the uterus have distinct combined respiratory and metabolic acidosis as compared to normal calves. Their blood gas and acid-base parameters have been found to be:[4,6]

pCO_2 = 72.7 mm Hg
pO_2 = 18.0 mm Hg
pH = 7.039
Base deficit = 10.6 mmole/L

The metabolic component lasts for six hours before compensation by the calf is complete.

Corticosteroids play a role in this acidosis. Cortisol and cortisone are at high levels at birth due to increased fetal adrenal activity which precedes and is part of the cascade of events leading to parturition.[7,8] A negative linear relationship exists in the neonate between pH and plasma cortisol.[9] Added fetal stress of dystocia will raise its cortisol level and consequently play a role in worsening the acidosis.

Calves delivered by caesarian section before physiologic parturition takes place suffer two problems. Their acidosis is worse than that of naturally delivered calves,[5] and they suffer worse respiratory distress from hypoxia and from a low ratio of lecithin to sphingomyelin, making a defective surfactant system in the lungs.[10]

The type of delivery will often determine the steps a veterinarian will take in his attempt to keep a calf alive. The dystochic calf is frequently in a life-compromising situation. Life support techniques may make a difference for many dystochic calves. Calves born by a natural parturition are not to be ignored as good husbandry techniques are vital to their continued health and well-being.

Post-Delivery

Once the calf is on the ground a prompt, quick evaluation of its viability must be made. All calves are slightly acidotic when born. This will cause some central depression which nets arteriolar constriction and decreased pulmonary circulation. How depressed is the calf? How dyspneic? Is there muscle tone or are the muscles flaccid? Are reflexes present (fore and hind limbs, swallowing, corneal, anal)? Is the pulse one hundred or better? Is the calf alive (presence or absence of corneal opacity)? An evaluation of these signs will tell if therapy is needed and/or warranted.

Adaptation

The signs listed above will give an assessment of how well neonatal adaptation has progressed. The neonate must quickly adapt itself to its new extrauterine environment, overcome the stress of parturition, and solve the hypoxic crisis resulting from severance of the umbilical cord. Respiration is the most urgent adaptation that must be made; all the other adaptations the neonate makes hinge upon it. Although it has been shown that there are intrauterine ventilatory efforts, they are weak because the stimuli to the central nervous system are buffered by the intrauterine environment. The first breath marks the end of fetal life and the beginning of the post-natal period. The muscles of respiration must create a large negative intrathoracic pressure to pull air into the lung and move the fetal lung liquid through the airways. The lung liquid is primarily removed by pulmonary capillaries and lymphatics. Little fluid drains externally. The decrease in pulmonary vascular resistance decreases the pulmonary artery pressure. Circulatory pathways can then change by reversal of blood flow through the ductus arteriosus, its closure, the closure of the foramen ovale, and the sealing of the umbilical arteries by clots. Thermoregulation must begin or hypothermia will result. Neonatal calves must rise and suckle shortly after birth to establish a successful neonatal-maternal bond and to obtain passive immunity from colostrum to change its agammaglobulinemic state of birth.

In most cases the events of delivery and adaptation proceed smoothly. A veterinarian can relieve dystocia but is unable to correct an adaptational cardiovascular irregularity. He can intervene successfully with many respiratory inadequacies. The veterinarian can also assist or advise the herdsman concerning avoidance and treatment of cold stress, on the necessary feeding of colostrum, and other husbandry techniques.

Respiratory Support

With the birth of the calf complete, one should express mucus from the nose and mouth with the thumbs pressed on the bridge of the nose and fingers in the intermandibular space. The hands are then slid from eyes to muzzle.[11] Stimulation of respiration can be accomplished by applying some cold water to the calf's head, rubbing the skin, tickling the nasal mucosa with straw, or tapping the thorax dorsocaudal to the heartbeat on the phrenic nerve. Administration of doxapram, pentylenetetrazol, or nikethamide can also be performed.

All of the above merely increase central nervous system stimulation and depend on the calf's innate ability to generate sufficient negative inspiratory pressure to inflate the lungs. A weak, depressed calf will not respond to added central nervous system stimulation.

If the calf is truly asphyxic, don't delay ventilatory support by using the above methods, but move to one of the following techniques:

(1) artificial respiration—With the calf in lateral recumbency, extend the neck, hold the mouth open, grasp the uppermost humerus and costal arch, and lift. Little aspiration is noted at first as the lungs are atelectatic. Continue to raise and lower the humerus until breathing begins or another technique is employed.

(2) intubation—This is the only way to insure lung inflation in a depressed calf. The best method of endotracheal intubation is the use of a long-bladed laryngoscope to displace the tongue and epiglottis ventrally and illuminate the laryngeal opening. Blind intubation by externally palpating the trachea and larynx with one hand and manipulating the endotracheal tube with the other requires more time but is possible. Endotracheal tubes 6.0–10.5 mm internal diameter will fit most calves. A 7 mm tube with a large inflatable cuff will fit all but the very small calf.[12] Be sure the tube is in the trachea; placement of the tube in the esophagus is easily done and easily checked by tracheal palpation.

(3) ventilation by Hudson Demand Valve[a]—The goal of this support is to inflate the calf's lungs in order that circulatory adaptations occur. This device will perform this operation without the danger of high gas pressure causing lung damage. It can deliver oxygen via an endotracheal tube to a maximum pressure of 39 mm Hg. The valve works by either manually pressing a button on the valve housing to supply gas flow or by demand of the calf after spontaneous ventilation begins. The apparatus is small, consisting of the demand valve and hose, an oxygen tank, a two-stage regulator, and an endotracheal tube. It would be an asset not requiring much room in a clinic or a vehicle.

(4) ventilation by ambu resuscitation bag—This device cannot deliver the pressure one can get from compressed gases. Manual ventilation with either room air or oxygen can be achieved. These bags require little space needing only an endotracheal tube or face mask for operation.

(5) ventilation by Vetaspirator[b]—This device will provide both suction and positive pressure. The attempt at suction of fluid from neonatal calf lungs is of little benefit. As pointed out earlier, the majority of lung fluid will be removed by lymphatics and pulmonary capillaries. The primary goal here is adequate lung inflation. The positive pressure side of this equipment will accomplish the lung inflation. The apparatus is somewhat bigger than the demand valve. It is also more complex to operate. An oxygen tank, two-stage regulator, hose, and face mask or endotracheal tube are required for operation.

(6) oxygen rich environment—This can be provided by a cage, tent, nasal cannulas, or face mask and an oxygen tank with regulator. This technique will not expand the lungs but will aid a struggling, breathing calf.

Following the initiation of ventilation by the calf, medications may be warranted. Calves needing cardiovascular volume support will benefit from intravenous administration of Ringer's or 5% dextrose in water. Acidotic calves with base deficits greater than 10 mmole/L develop severe respiratory distress. Morrow's suggested technique of administration of sodium bicarbonate and glucose intravenous solutions does alleviate the metabolic acidosis but makes the respiratory effects worse.[13] Treatment with sodium bicarbonate and glucose solutions were no better than saline for survivability of acidotic calves. This lack of success has been ascribed to possible lung lesions before treatment (atelectasis, alveolar emphysema, hyaline membranes, interstitial edema), brain hemorrhages, and/or in-

[a]Hudson Demand Valve, Hudson Oxygen Therapy Sales Company, Wadsworth, Ohio.

[b]Vetaspirator Oxygen Resuscitator, Veterinary Specialties, Inc., Cedar Rapids, Iowa.

tracellular acidosis.[14]

Umbilical Stump

Severe the cord 10 cm from the body with a sterile scissors. Don't dress the stump unless there is a farm history of omphalitis, joint ill, etc. If conditions merit treating the stump, press out the jelly found within the cord and paint it with copper sulfate, 20% tincture of iodine, 5% lysol, or an aerosol antibiotic.[15]

Colostrum

The herdsman must be made aware of the value of colostrum and its proper administration. The worth of this practice cannot be stressed enough. Colostrum provides an energy source and important immunoglobulins. The capacity of the fetus to respond to antigens is gradually developed to arrive at immunocompetency at birth. The calf fetus can respond to some antigens at 132 days and others not until term. Neonates are susceptible to disease because of the unprimed state of their immune system, not due to incapacity. The hazards associated with the failure of passive transfer of immunoglobulins to the agammaglobulinemic neonate are severe. A positive relationship has been found between low blood serum immunoglobulin levels and subsequent mortality in calves.[16] Low levels of immunoglobulins are correlated with calf enteritis, omphalitis, and hyperthermia. Given this relationship with disease and mortality, proper colostrum ingestion is important for all calves.

In one study, eighty percent of normal dairy calves stood within ninety minutes and had suckled within eight hours. Calves rarely suckle from a recumbent dam.[17] Force-feeding two liters of colostrum within one hour of birth is one guideline presented.[18] Two liters per calf at first feeding is a commonly suggested amount, while the time of feeding has sometimes been increased to within sixteen hours after birth.[19] This latter guide is questionable for two reasons: closure of the calf's intestine to immunoglobulin absorption (which will be discussed later) and the decrease in quality of colostrum which begins at nine hours after parturition. Consequently, tube or bottle administration is recommended as soon as possible after birth.

The age of the calf when fed colostrum and the amount fed are very important in the administration of colostrum or a blood-derived substitute. These two factors determine the serum immunoglobulin concentration of the post-colostral calf. With increasing age, there are fewer intestinal epithelial cells responsible for colostral immunoglobulin transfer. These enterocytes are capable of pinocytotic activity, colostral uptake, and transmission of colostral constituents into circulation. As these cells progressively decrease in number, there is a concomitant decrease in absorption rate in both the initial period after feeding and in succeeding periods.[20] All of the three activities: pinocytosis, uptake, and/or transmission to circulation are affected. This process, termed "closure," begins at twelve hours. Mean closure finish time is twenty-four hours.[21] Passive immunoglobulin transfer occurs in six to twenty-four hours after feeding.[22] There is no immunoglobulin absorption from the gut after forty-eight hours. Production of the calf's own immunoglobulin begins at a very low level at seventy-two hours.[23] Two liters of colostrum is optimum to feed the average Holstein calf from birth to sixteen hours. After that time, less should be fed to avoid immunoglobulin A and immunoglobulin M absorption inhibition due to excessive amounts.[19]

Earlier absorption rates and maximum absorption of colostral immunoglobulin were greater in calves that suckled their dams than in those bottle-fed. Some labile factor is transferred to the calf in the dam's colostrum acting as a messenger to stimulate a burst of pinocytotic activity.[24] Artifically feeding calves colostrum in the presence of their dam increased blood serum immunoglobulin levels.[16] Despite this, bottle feeding may be preferable because it allows confidence in the amount of colostrum ingested and when it occurs.

Calves with acidosis ($pH < 7.15$) have been shown to ingest less colostrum and have subsequent lower immunoglobulin levels. Normal calves fed the same amount as was ingested by the acidotic calves (smaller than normal) had higher immunoglobulin levels.[25] The effect of corticosteroids on dam production and calf absorption of colostral immunoglobulins is clouded. It has been shown that induction of parturition by corticosteroids decreases immunoglobulin G transfer to colostrum[26] and decreases the efficiency of immunoglobulin absorption by calves born to such cows.[27] Corticosteroids, natural or introduced, in normally delivered calves have been shown to cause no reduction in immunoglobulin absorption.[28]

Cold stress has been shown to delay onset and significantly decrease the rate of absorption of colostral immunoglobulins. Cold stress also affects calves adversely by causing loss of body heat by conduction and evaporation when their skin and haircoat are wet, in addition to the normal loss by radiation and convection. Calves are born wet with amniotic fluid and are extremely susceptible to chilling. Normal thermoregulation causes shivering and the catabolism of brown fat stores. In severe weather and/or housing conditions, calves' natural systems can't overcome cold stress. Thermoregulatory mechanisms of calves will return, so a heat lamp and/or a warm room is justifiable to augment their thermoregulatory systems.[29] If the dam doesn't lick her neonatal calf dry, sprinkling bran on the calf will help coax her along. Calves to be removed from their dams at birth should be rubbed vigorously with straw or sacking. The dam licking the anal region of the calf is stimulation for passage of meconium, the debris accumulated during fetal life. Colostrum feeding prolongs the time of defecation of meconium. Contrary to the idea of colostrum being a purgative, the more colostrum fed, the longer before meconium passage.[15]

To define good quality colostrum, a number of variables must be considered. Advice from the veterinarian concerning what colostrum to freeze for future use is of benefit to the stockman. Colostrum is better from a cow without any or only a little prepartum colostral loss. A cow that delivers prematurely has inferior quality colostrum. The herdsman who wants to freeze colostrum should pick cows that are older, have been on his farm a long time, have no mastitis, that calve in the fall, have greater parity, and delivered normally, rather than caesarian.[30] The teats should be washed before the calf suckles or the colostrum milked out to remove *E. coli* and *Salmonella spp.* that arrived there with feces or vaginal discharge.[1] Colostrum may be frozen for some time. Reports vary on successful storage times ranging from nine months to fifteen years. Freezing single dose aliquots is suggested for ease of administration. Thawing should be neither fast nor accomplished in a microwave. A natural thaw of the container placed out on a counter is best. The colostrum can be given while between room and body temperature. Colostrum at room temperature does not decrease intake nor blood serum immunoglobulin levels when compared to body temperature administration.[16]

For a situation where no colostrum is available for calves, several substitutes can be used. They can be given bovine plasma, serum, or whole blood. The dam has approximately 2 g/dl gammaglobulins and the desired level for the calf is the same. Blood volume is eight percent of body weight. If packed cell volume is thirty percent, then the blood is seventy percent fluids or serum. Give the calf one serum volume of serum or plasma intravenously rapidly through a fourteen gauge needle. Whole blood should be given over several hours.[31] Oral administration of plasma or serum can also be of benefit if the calf can still absorb the immunoglobulins from his intestine. An oral colostrum substitute given three times per day consists of:

a whipped egg in 300 ml of water,
½ teaspoon of castor oil, and
600 ml of whole milk.

Egg white is antibacterial for some *E. coli.*[15] This is not intended to replace colostrum but is a mixture to use when nothing else is available. There are no immunoglobulins in it.

Conclusions

Neonatal calf care is economically important and becoming more so. The veterinarian can assist in reducing calf mortality, particularly in respiratory support in asphyxic calves. Advice on herdsman husbandry will also aid in keeping calf mortality and morbidity low. The importance of colostrum, its proper dosage and administration, and the use of substitutes or frozen colostrum are ideas to stress to the cattle raiser. Keeping calves dry will reduce the weak calf syndrome induced by cold stress. Breeding programs to reduce the occurrence of dystocia will reduce the incidence of asphyxic, acidotic calves.

References

1. Hoffman B, Mason IL, Schmidt J: *Calving Problems and Early Viability of the Calf.* The Hague, Martinus Nijhoff, 1979.
2. Dennis SM: Low viability of calves at birth. *Vet Ann* 21:63–73, 1981.
3. Schlerka G, Petschenig W, Jahn J: Blood gases, acid-base relationship, electrolytes, some enzymes, and other constituents of blood from newborn calves. I. Blood gases and acid-base relationship. *Dtsch Tierarztl Wschr* 86:95100, 1979.
4. Mauer-Schweizer H, Wilhelm U, Walser K: Blood gases and pH value up to 24 hours after birth in calves by caesarian section. *Berl Munch Tierarztl Wschr* 90:215–218, 1977.
5. Eigenmann UGE, Grunert E, Koppe R: The delay-

ed asphyxia of calves. *Berl Munch Tierarztl Wschr* 94:249-254, 1981.

6. Mauer-Schweizer H, Walser K: Acidosis and clinical status in calves with asphyxia. *Berl Munch Tierarztl Wschr* 90:364-266, 1977.
7. Cabello G: Neonatal changes in plasma levels of cortisol, cortisone, and aldosterone in the calf. *Bio Neonate* 36:35-39, 1979.
8. Massip A: Relationship between the type of delivery and the acid-base and plasma cortisol levels of the newborn calf. *Brit Vet J* 136:488-491, 1980.
9. Massip A: Relationship between pH, plasma cortisol, and glucose concentration in the calf at birth. *Brit Vet J* 136:597-601, 1980.
10. Jahn D: Causes of dyspnea in prematurely born calves with reference to the surfactant system. *Inaugural-Dissertation Tierarztl Hochschule Hannover,* 1982.
11. Drost M: Perinatal care of the calf in Morrow DA (editor): *Current Therapy in Theriogenology.* Philadelphia, W.B. Saunders Co, 1980, 274-275.
12. Brunson DB: Ventilatory support of the newborn calf. *Comp Cont Ed* 3:S47-52, 1981.
13. Eigenmann UGE, Rudiger B, Schoon HA, Grunert E: Sodium bicarbonate and glucose administration for asphyxial calves. *Dtsch Tierarztl Wschr* 89:217-260, 1982.
14. Falk B: Treatment of dyspnea in newborn calves by intravenous infusion of sodium bicarbonate and glucose. *Inaugural-Dissertation Tierarztl Hochschule Hannover,* 1982.
15. Roy JHB: *The Calf.* London, Butterworth's, 1980.
16. Fallon RJ: The effect of immunoglobulin levels on calf performance and methods of artificially feeding colostrum to the newborn calf. *Ann Recherches Vet* 9:347-52, 1978.
17. Suzuki S, Nakajima M, Nakamura Y: Natural sucking behavior in dairy calves during the first twenty-four hours after birth. *Jap J Zoo Sci* 50:778-781, 1979.
18. Blackmer PF: Dairy calf health management in Howard JL (editor): *Current Veterinary Therapy, Food Animal Practice.* Philadelphia, W. B. Saunders Co, 1981, 135-140.
19. Stott GH, Marx DB, Menefee BE, Nightengale GT: Colostral immunoglobulin transfer in calves, III. Amount of absorption. *J Dairy Sci* 62:1902-1907, 1979.
20. Stott GH, Marx DB, Nightengale GT: Colostral immunoglobulin transfer in calves, II. The rate of absorption. *J Dairy Sci* 62:1766-1773, 1979.
21. Bush LJ, Staley TE: Absorption of colostral immunoglobulins in newborn calves. *J Dairy Sci* 63:672-680, 1980.
22. Fischer EW: Discussion of Congress' paper by Watt JG. *Vet Record* 77:1484-1486, 1965.
23. Devery JE, Davis CL, Larson BL: Endogenous production of immunoglobulin IgG1 in newborn calves. *J Dairy Sci* 62:1814-1818, 1979.
24. Stott GH, Marx DB, Menefee BE, Nightengale GT: Colostral immunoglobulin transfer in calves, IV. Effect of suckling. *J Dairy Sci* 62:1908-1913, 1979.
25. Eigenmann UGE, Zaremba W, Luctgebrune K, Grunert E: Investigations on the colostrum intake and absorption of immunoglobulins by calves with and without acidosis. *Berl. Munch Tierarztl Wschr* 96:109-113, 1983.
26. Brandon MR, Husband AJ, Lascelles AK: The effect of glucocorticoid on immunoglobulin secretion into colostrum in cows. *Aust J Exp Biol Med Sci* 53:43-48, 1975.
27. Husband AJ, Brandon MR, Lascelles AD: The effect of corticosteroid on absorption and endogenous production of immunoglobulins in calves. *Aust J Exp Biol Med Sci* 51:707-710, 1973.
28. Stott GH: Immunoglobulin absorption in calf neonates with special considerations of stress. *J Dairy Sci* 63:681-688, 1980.
29. Olson DP, Papasian CJ, Ritter RC: Effects of cold stress on neonatal calves, I. Clinical condition and pathological lesions. *Can J Comp Med* 44:11-23, 1980.
30. Frerking H, Aeikens T: About the importance of colostrum for the newborn calf. *Ann Recherches Vet* 9:361-365, 1978.
31. Hoffsis G: Saving the valuable calf in *Proceedings, Tenth AABP Convention,* pp 119-124, 1977.